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THE SURGICAL MANAGEMENT OF BRAIN ABSCESS

By Alfred W. Adson, M.D.,
and
Winchell McK. Craig, M.D.
of Rochester, Minn.
FROM THE SECTION OF NEUROLOGIC SURGERY, THE MAYO CLINIC, ROCHESTER, MINNESOTA

The management of brain abscesses still appears to be in a state of confusion. Many surgeons are in doubt when to drain an abscess and are uncertain concerning the procedures to use. We believe a review of our experiences at the clinic might therefore offer some assistance in the management of such abscesses.

Cerebral abscess results from intracranial invasion of pyogenic organisms from acute or chronic infection of the middle ear, or frontal or accessory nasal sinuses, from compound penetrating injuries to the skull, and from infected emboli associated with such chronic suppurative lesions as osteomyelitis and empyema or purulent bronchiectasis. A history of initial infection with subsequent extension to the brain usually can be elicited. This is characterized by symptoms of meningitis or localized encephalitis, with or without an elevation of temperature, which in progressing produces signs of intracranial pressure. The three stages in the development of a cerebral abscess are (1) the initial stage of localized meningitis and encephalitis, with spontaneous recovery or progression to the second stage; (2) the quiescent stage, with encapsulation and liquefaction necrosis, and (3) the terminal stage, with recovery or death. Recovery may occur spontaneously with inspissation or resolution of pus, or it may occur following surgical drainage. Death ensues as a result of rupture of the abscess into the subarachnoid spaces and ventricle, or into adjacent brain tissue, with further extension of the lesion. The most expedient time for surgical treatment is during the second, or quiescent stage, and the success of the procedure depends on adequate and thorough drainage.

The diagnosis and treatment of brain abscess has been gradual in evolution, the earlier pathologists distinguishing between purulent and non-purulent encephalitis. Recognition of the etiological factors associated with abscess of the brain by Toynbee[33] in England in 1855, and by Biermer in Germany in 1867, opened a new avenue of investigation. Clinical investigation was begun by Wernicke,[35] whose pioneer work on aphasia augmented our knowledge of the diagnostic possibilities and the peculiar symptoms produced. Barker,[7] Horsley,[23] and Ballance[6] were among the pioneers who attempted to deal with brain abscess by means of surgical interference. In a classical contribution, Macewen,[29] in 1893, presented a summary of the essential features in diagnosis and treatment advocated at that time. Warrington,[34] in discussing the etiological factors of brain abscess, divided the methods of

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invasion of the cranial cavity by the infecting organism into two major groups, the more common including foci in contiguous structures. This group included infections following injuries to the skull, in a great many cases resulting from osteomyelitis developing from purulent infection in the mucous membrane lining the cavities in the temporal bones or in the accessory nasal sinuses. The second group of important etiological factors he designated as infections of the brain through the blood-stream from some distant site of suppuration, resulting in metastatic or hæmatogenous brain abscess. In this classification were included osteomyelitis of the long bones, bronchiectasis, pulmonary abscess and empyema, and, in rare instances, pericarditis, infective endocarditis, and so forth.

TABLE I

Evans' Classification of Methods of Invasion

A. Direct extension		
1. Otitis media and mastoiditis		109
a. Purulent thrombosis of the lateral sinus	37	
b. Osteomyelitis of tympanic wall	28	
c. Spread via internal auditory meatus	1	
d. Spread along the adventitial spaces of perforating blood-vessels	43	
2. Suppuration in the nasal cavity and accessory sinuses		12
a. Osteomyelitis of the sinus wall	7	
b. Spread along perineural spaces	3	
c. Spread along veins	2	
3. Following trauma to skull		8
4. Invasion of the brain by malignant growths		2
B. Hæmatogenous spread		
1. Metastatic brain abscess resulting from intrathoracic suppuration		22
a. Chronic bronchiectasis	17	
b. Empyema	5	
2. Metastatic brain abscess resulting from extrathoracic suppuration		24
C. Method of spread doubtful		2
D. Source, and therefore method, of spread doubtful or unknown		15
Total		194

Evans,[17] in a study of 194 cases of brain abscess at necropsy, discussed the etiology under headings depending on whether infection was transmitted to the brain from an adjacent or distant primary focus (Table 1).

Suppurative otitis media is the infection which most frequently leads to intracranial complications, and this results from direct extension of the infective process. Evans stated that 83.2 per cent. of all brain abscesses in his series were the result of direct extension from the middle ear and antrum. Macewen discovered that an abscess may occur independent of a visible tract of inflammation, the organism being conveyed by direct vascular connections. This is either due to an infective thrombosis spreading through the veins into the pia mater or brain or to a reversal of the blood-stream with carrying of portions of the infected thrombus inward. Although chronic otitis media prevails as a causative factor, acute otitis may lead to brain abscess when the infection is a virulent one.

In an anatomical discussion of the regions most susceptible to attack by caries and necrosis Macewen gave precedence to the mastoid region; he placed the roof of the tympanum and antrum second, the sigmoid groove third, the posterior wall of the external auditory canal fourth, the tympanic floor with the posterior wall of the carotid canal fifth, and the petrous portion of the middle ear, including the structures, sixth.

Although brain abscess may occur at any age, it is more common among adults. According to Holt[22] brain abscess is rare in very young children, although Evans reported twelve cases in which the patients were less than five years of age. A survey of the literature reveals that the situation of a brain abscess of otitic origin is as a rule in the temporosphenoidal lobe or in the cerebellum, although a few cases have been reported in which the abscess was found in the occipital and in the frontal lobes. In a series of 645 cases collected by Heiman,[21] 395 abscesses were in the temporosphenoidal lobe and 196 in the cerebellum. Evans stated that brain abscesses, regardless of cause, occur about twice as often in the cerebrum as in the cerebellum, but that when those caused by distinct foci were eliminated, the ratio changed and the frequency with which the lesion was situated in the cerebrum was found to be markedly diminished.

ETIOLOGY AND PATHOLOGY.—Streptococcus pyogenes and Staphylococcus pyogenes aureus, according to Macewen, are the organisms most frequently demonstrated by the microscope and isolated by culture, although besides these organisms pneumoccoci, hæmolytic streptococci, or, though rarely, colon bacilli, may be the infective organism.

AVENUES OF INFECTION.—Brain abscesses result from three avenues of cerebral infection: inoculation, extension by contiguity, and embolus.

Inoculation of the brain with septic organisms results from compound, comminuted fractures of the skull, puncture wounds extending through skull into the brain, and gunshot wounds. Fortunately not all wounds result in the formation of abscess. The type and virulence of the organism, as well as natural immunity, play important parts in determining whether or not abscess will develop. The emergency surgical treatment, immediate removal of infected, necrotic brain tissue, may prevent the development of an abscess.

Extension of infection by contiguity is responsible for the largest number of brain abscesses. They follow furuncles, lacerations, and cellulitis of the scalp, and osteomyelitis resulting from infected wounds and extension from nasal sinus and mastoid infections. Again the virulence of the organism and the natural immunity of the patient to the invading organism are important factors in determining whether or not a cerebral abscess will develop. However, the efficiency of the cerebral circulation is just as important a factor as are virulence and immunity, since one rarely sees a cortical abscess when arterial circulation is adequate. Infections may involve the scalp, skull, and meninges without giving rise to a cerebral abscess. This phenomenon, we believe, is due to the fact that meningeal reaction has not resulted in the thrombosis of veins extending into the white substance of the brain. Local infection of meninges may produce symptoms of meningitis with or without organisms in the spinal fluid.

Cerebral abscesses are invariably situated below the cortex. Occasionally a stalk can be seen to extend from the meninges to the abscess, but more often than not no trace of invasion can be demonstrated. The explanation for this, we believe, is due to the fact that a communicating vein has become thrombosed and has served as a channel for the organism to invade the white, poorly-vascularized brain tissue. The stalk, if present, represents the zone of reaction about the infected vein. Following the development of the infected thrombosed vein there is a localized area of encephalitis. This process extends by thrombosing more capillaries and vessels until immunity and reaction limit the extension. The centre of the process disintegrates, liquefies, and becomes pus. Proliferation in the peripheral zone results in fibrous formation and encapsulation of the pus.

We believe that infections may travel through thrombosed veins and diploë for long distances and produce abscesses without giving rise to local osteomyelitis during their course.[3] This is substantiated by the fact that abscesses result from infections of the frontal sinus, antrum, and superior maxilla. The reason that cerebral abscesses do not always follow osteomyelitis of the flat bones of the calvarium, frontal sinusitis, disease of the middle ear, or mastoiditis is due, we believe, to the fact that venous thrombosis becomes limited and a collateral venous circulation is maintained.

A single embolic abscess is relatively rare. Multiple abscesses follow pulmonary disease, particularly bronchiectasis. These may also represent part of a general septicopyæmia. The infection associated with multiple abscesses is usually so virulent and overwhelming that death results before encapsulation takes place.

Bagley[4, 5] in a comprehensive pathological study of brain abscess, divided the avenues of infection into four groups. Hassin,[20] in a study of the histopathology of brain abscesses, classified them under the two general headings, acute and chronic, making his differentiation on the formation of a fibrous wall consisting of three layers. He stated that in acute abscess, in which the reaction of the brain tissue, in diffuse suppuration, has not had sufficient time to become encapsulated, there is a tendency toward total destruction of ganglion cells with partial destruction of the nerve fibres and the glial cells (observed also by Cone[12]) and toward progressive vascular changes consisting in hyperæmia and thickening with infiltration of the vessel walls. In chronic abscess, wherein the process has been of sufficient length to allow the formation of a capsular wall, the changes are characteristic. The fibrous capsule derives its structure from the lymphocytes of the blood by formation of fibroblasts which continue to accumulate until the local area of infection is surrounded and walled off.

Penfield[32] stated that the wall of an abscess begins to form in the first week but that it is not firm enough to offer appreciable resistance to an exploratory needle until two to three weeks have passed. The course without drainage depends on the nature and virulence of the organism. If an insufficient wall or capsule is not formed, spreading encephalitis with œdema of the brain may quickly terminate the patient's life.

Symptoms.—The symptoms of cerebral abscess vary with the different stages of the disease and vary according to the systemic reaction, the degree of increased intracranial pressure, and the situation of the abscess.[31]

Initial Systemic Symptoms.—Patients with temporosphenoidal or cerebellar abscess invariably give a history of chronic otitis media or acute otitis media which suddenly stops draining or of the development of a suppurative mastoid. Abscesses in the frontal lobe are preceded by acute colds, frontal sinusitis, pansinusitis, and osteomyelitis of the frontal bone. The sudden increase in septic reactions plus cerebral involvement of patients with cranial wounds, osteomyelitis of the skull, and general pyæmic infections, including bronchiectasis, suggest the development of a cerebral abscess. Patients become apathetic and an increase in temperature develops; the temperature remains high for the first few days but soon takes on the septic, steeple-like curve. The leucocyte count jumps to 20,000 or more per cubic millimetre. Examinations of spinal fluid disclose an increase of pressure, increased protein, and an increase of lymphocytes.[37] If an active suppurative meningitis develops, polymorphonuclear leucocytes appear in large numbers, causing the fluid to become cloudy. The active organisms also are usually identified.

Initial Cerebral Symptoms.—Headaches and vomiting appear early among the symptoms. Irritability, alternating with a stupor and with rigidity of the neck muscles, are signs of increased intracranial pressure and meningeal irritation. The pulse is full, bounding, and slower in rate. Choked disks and retinal hæmorrhages likewise appear when the normal flow of cerebrospinal fluid has been disturbed.[8, 28]

Initial Localizing Signs.—The localizing signs depend on the size and situation of the abscess. A *temporosphenoidal abscess* can produce homonymous field defects, homolateral palsy in the distribution of the sixth and third cranial nerves, and contralateral anæsthesia, paralysis, and pathological reflexes. An *abscess in the frontal lobe* may attain considerable size without producing localizing symptoms.[24] However, we have observed that the initial swelling about one eye is a good diagnostic sign of the lobe affected since the abscess is usually on the same side. Osteomyelitis of the frontal bone invariably extends to one or the other sides of the median line, which suggests the lobe involved. Although paralysis may not be present, reflexes may be exaggerated on the contralateral side. When in doubt, one is justified in carrying out ventricular study and ventriculography. *Cerebellar abscesses* produce ataxia of the cerebellar type, plus nystagmus and reduction of homolateral reflexes. Hiccoughs and rigidity of the neck further indicate a lesion of the posterior fossa. Those *miscellaneous abscesses* that involve the brain stem and pons present bilateral pyramidal signs and bilateral palsy in the distribution of cranial nerves. Those developing from infected wounds are situated in the vicinity of the infection, and those developing from nasal sinuses other than the frontal sinus are situated in the lower half of the frontal lobes.

We have seen *temporosphenoidal abscesses* and one *cerebellar abscess* produce homolateral pyramidal symptoms with partial contralateral pyramidal symptoms. The symptoms all disappeared following drainage of the abscess. The homolateral pyramidal symptoms were undoubtedly due to partial displacement of the hemisphere by the abscess to the opposite side to such an extent that the crus cerebri was pressed and notched by the opposite margin of the tentorium at the incisura tentorii.

Secondary Symptoms of Quiescent Stage.—The pathological process, during the initial stage (up to three weeks), represents a battle between destructive and reparative forces. The secondary quiescent stage, second to fifth week, represents a partial victory for the forces of repair, since the abscess no longer enlarges and the pus is becoming more securely confined by a fibrous capsule. The œdema disappears and circulatory disturbance, peripheral to the capsule, is slowly repaired. The recovery will continue up to five weeks, with large abscesses, when it apparently comes to a standstill. During this quiescent stage the symptoms of sepsis subside, and the temperature and leucocyte count recede to normal or just slightly above normal. Headaches improve, mental reactions are faster, and the cerebrospinal fluid count returns to normal. Localizing symptoms likewise subside, but in spite of the general improvement periodic headaches occur, and choked disks and field defects fail to disappear. Reflexes remain exaggerated and motor impairment persists. In cases of abscess of the frontal lobe, symptoms of euphoria or depression linger. The leucocyte count continues to rise to 12,000 or 14,000 per cubic millimetre. The patient has days when he is apathetic and refuses to eat, and prefers to lie in bed. If it were not for the history of the infection and the symptoms of the initial stage, it would often be difficult to distinguish those due to abscess from those produced by a cerebral neoplasm.

Symptoms of the Terminal Stage.—Symptoms may continue on from the quiescent stage for months until the patient is operated on through an erroneous diagnosis of brain tumor or dies from rupture of the abscess into the ventricular system or subarachnoid spaces.[19] Small abscesses heal spontaneously with disappearance of all symptoms. Symptoms of abscesses that have been drained surgically disappear unless important tracts and cortical centres have been destroyed by the inflammatory process. Mutilating operations likewise contribute to permanent injury of cerebral tissue. Epilepsy is a sequel to localized encephalitis and abscess, and it may appear in any case in which there has been a lesion in the frontal, temporal, or parietal lobes. The incidence of this condition may be lowered by proper and adequate drainage. Subsequent scar resection may offer some amelioration from this symptom.

Surgical Considerations.—Appropriate Time for Drainage.—It took us many years to learn that hasty emergency operations were futile and were accompanied by high mortality.[1] This, undoubtedly, was due to the insistence of colleagues that we do something as soon as a diagnosis of cerebral abscess had been made. Every cerebral abscess passes through a stage of encephalitis before encapsulation.[11] It is during this stage that the infection is virulent and is most easily disseminated. Some surgeons argue that unless the necrotic tissue is removed the patient will die. This is true in the occasional case, and cases have been reported in which aspiration of necrotic material was successful; more often than not, however, the infection is disseminated by surgical intervention and the patient dies from a fulminating, suppurative meningo-encephalitis. We believe the best procedure to employ during the acute stage is supportive treatment, rest in bed, high calorie diet, spinal drainage, ice bags to the head, frequent catharsis, moderate amounts of fluids, and, if the patient is comatose, occasional intravenous administration of hypertonic glucose.

Encapsulation takes place in two to four weeks. The process is an indication that immunity is being established. It is characterized by a fall in the leucocyte count from 20,000 or more to 12,000 or 14,000 per cubic millimetre. The temperature likewise returns approximately to normal, 100° F. or below. The cell count of the cerebrospinal fluid, if increased, returns to normal. The cerebral symptoms gradually subside, but seldom completely disappear until the abscess is drained. Choking of the optic disks, if present, may continue until optic atrophy results. When encapsulation and immunity have been established, thorough and continuous drainage is necessary to effect a cure without recurrence of the abscess.

SURGICAL TECHNIC

The ideal exposure of a cerebral abscess is one that allows the surgeon to enter the cranium through a clean field over the abscess. Exceptions to this rule are when one wants to avoid a scar in the frontal region or is compelled to pass through a zone of osteomyelitis to reach the abscess. In entering the skull through a clean field the site is chosen where the abscess is nearest to the cortex or will give the best drainage. A small incision, 5 centimetres in length, is made in the scalp (Fig. 1). A craniotomy 3 centimetres in diameter (Fig. 2) is usually large enough to afford ample exposure. The meninges and cortex are then sutured with interrupted stitches of catgut about the margins of the incision in the skull to prevent separation of the cortex from the dura (Fig. 3). A crucial incision is made in the dura to expose an area about 2 centimetres in diameter. The meninges and cortex are further sealed and glued together with the electrocoagulating current, about the margins of the incision in the skull, a suggestion of Learmonth's, to protect further against spread of pus over the cortex under the dura. The margins of the wound are covered with wet cotton strips. The cortex overlying the abscess is frequently œdematous and cyanotic in appearance.

A round-tipped brain cannula is used to locate the abscess. The resistance

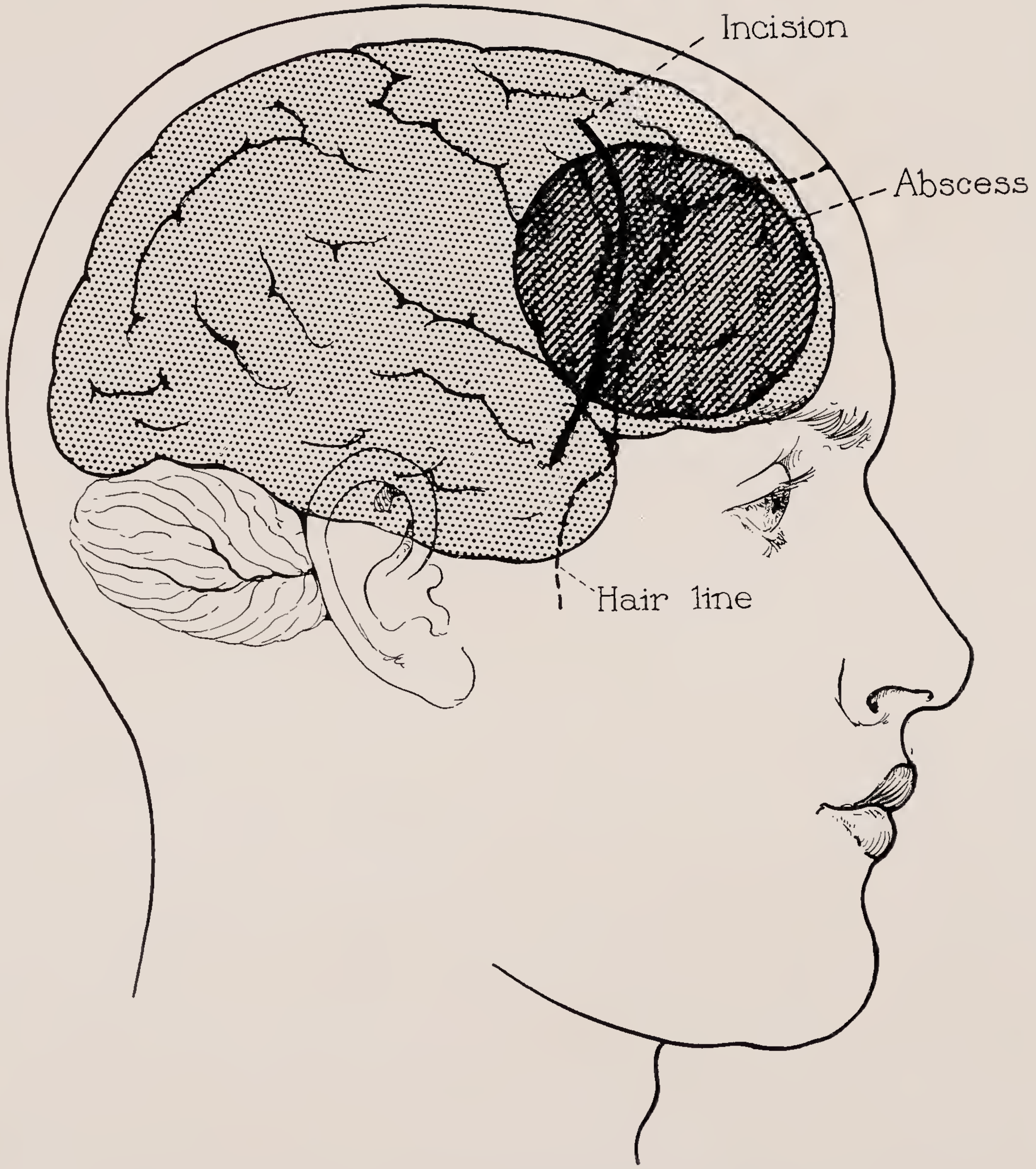

Fig. 1.—Incision in scalp for abscess in the right frontal lobe.

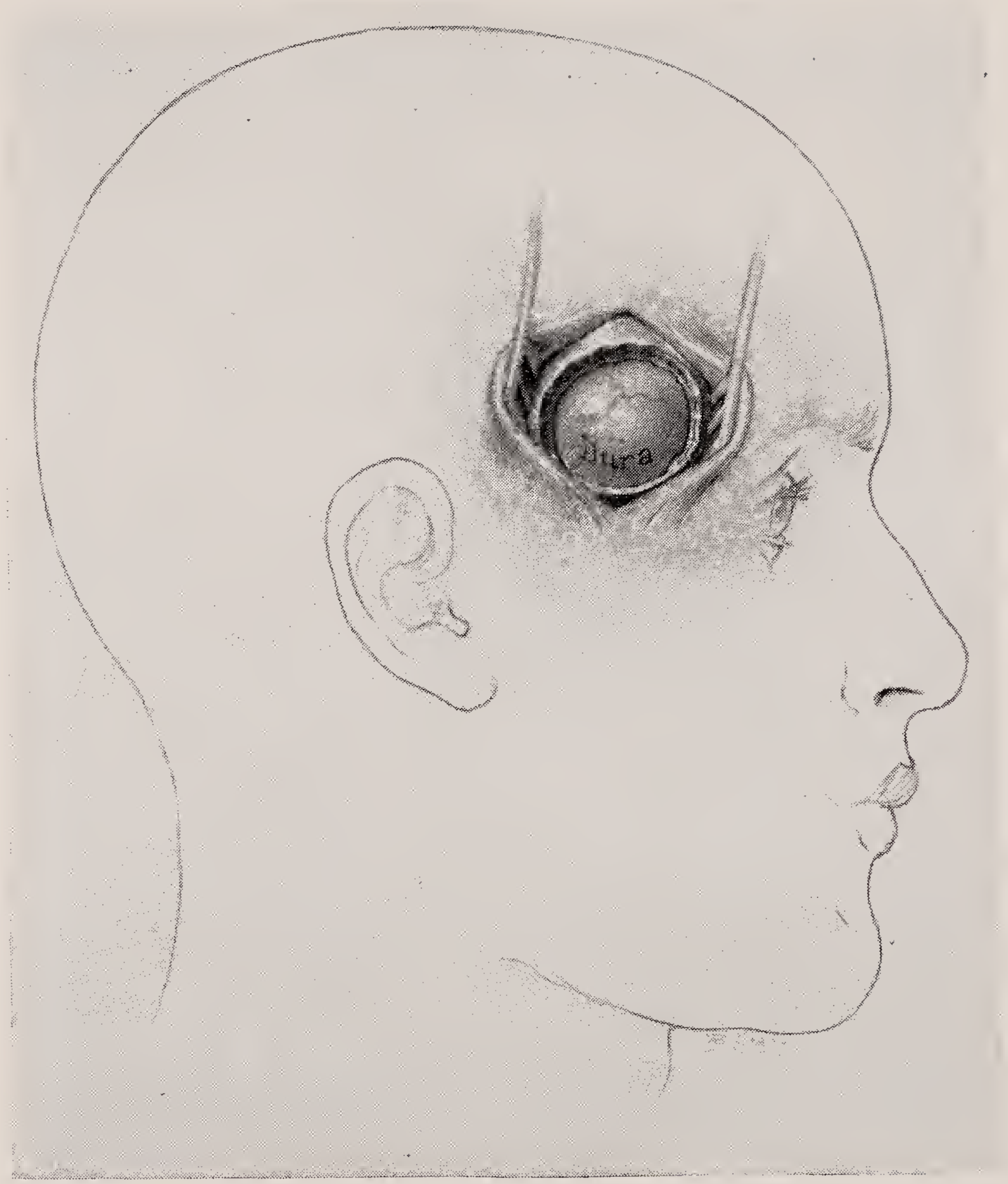

Fig. 2.—Craniotomy illustrating the dependent position for draining an abscess of the right frontal lobe.

of the capsule of the abscess gives one the impression that the cannula is being placed against a flexible, hollow, rubber ball. If the abscess has been there for a long time, the resistance may be so great that it is impossible to penetrate the capsule without incising it with a bistoury. Small abscesses may be overlooked, since the firm capsule is capable of deflecting the cannula unless the cannula is directed toward the center of the abscess. As soon as the cannula enters the abscess, the trochar is removed and a Luer's syringe with an intervening rubber connecter is attached to the cannula. The pus is gently aspirated, and the cavity is cleansed with small quantities of normal saline solution (Fig. 4). The cavity of the abscess is explored by incising the cortex and capsule with an electrosurgical needle, using the cannula, which has been left in place, as a guide. The capsule is opened for a distance of 2 centimetres in order to insert an illuminating retractor. This makes intracapsular exploration possible, permits further cleansing of the cavity of the

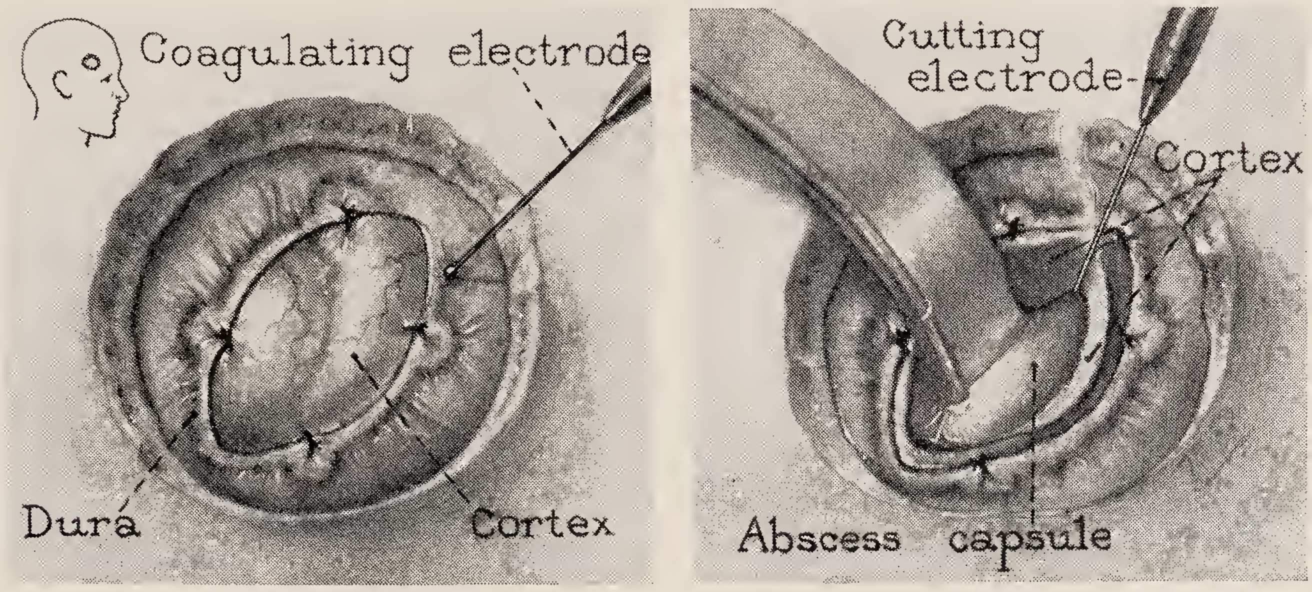

FIG. 3.—*Left.*—Procedure in sealing meninges to cortex with interrupted sutures of catgut and electrocoagulating needle. *Right.*—Incision of cortex exposing capsule of abscess.

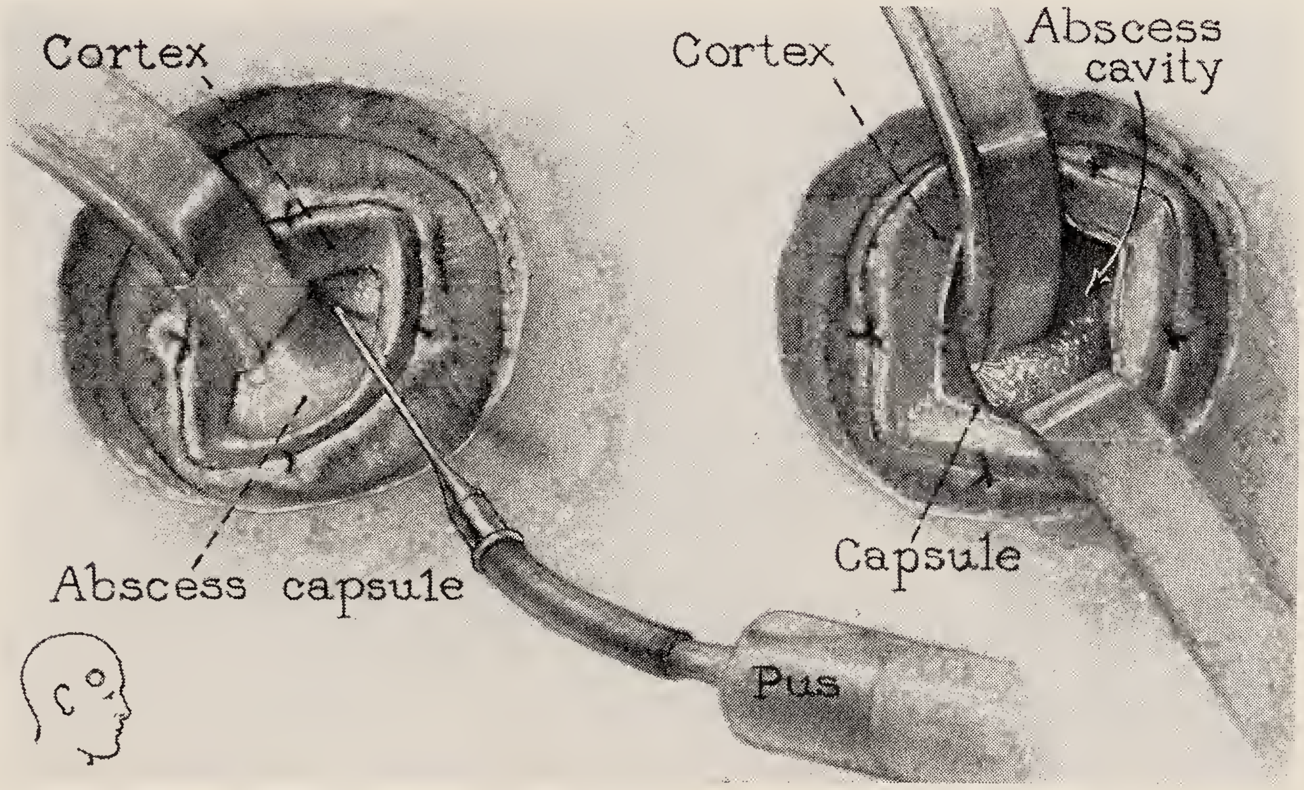

FIG. 4.—*Left.*—Partial aspiration of pus with cannula before incising capsule of abscess. *Right.*—Exploration of abscess and cleansing of abscess cavity.

abscess, and assures against overlooking communicating abscess pockets. During this procedure the retractor is used to hold the superficial wall of the capsule and the cortex against the skull.

To assure further against collapse of the cortex following drainage of an abscess, we fill the cavity about the two inserted tubes with loosely packed, iodoform gauze strips (Fig. 5). These continue to keep the capsule moderately distended, instead of allowing it to crumple down and give rise to loculated pockets within the capsule. The gauze strips are shortened daily until they are removed on the tenth day; this permits gradual collapse and

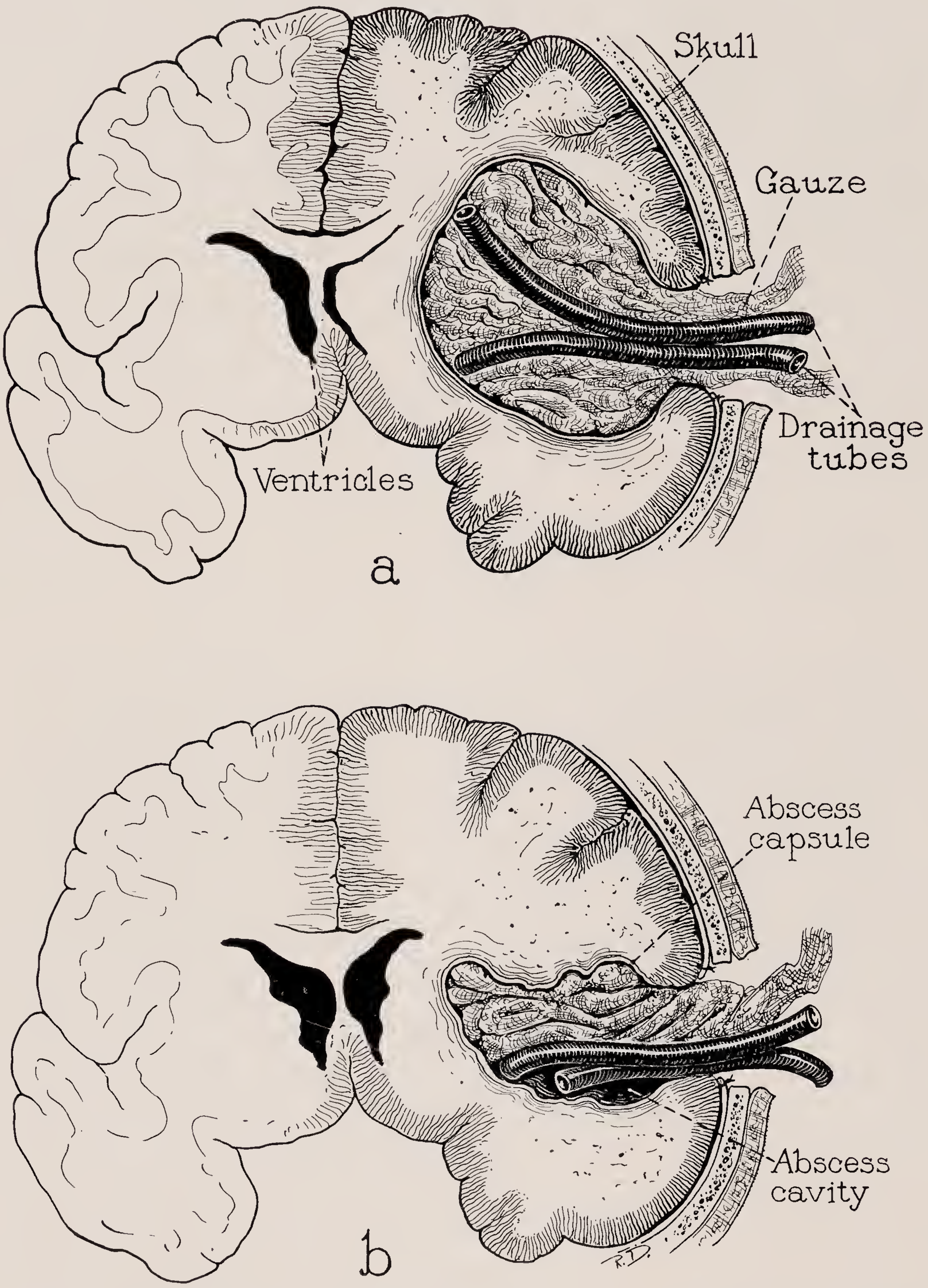

Fig. 5.—*a.*—Method of draining abscess cavity to assure adequate drainage and prevent pocketing of abscess cavity. *b.*—Gradual withdrawal of drainage material and resulting collapse of the abscess capsule.

contraction of the capsule and prevents recurrence of abscesses and cerebral fungi.

The two tubes are sections of number 18 catheters. They are left undisturbed until after the gauze has been removed. One is shortened on alternating days until removed on the twenty-first day; the other is shortened as the sinus closes in and forces it out, which requires from four to six weeks. At operation the tubes are fastened to the skin to prevent accidental removal during the daily withdrawal of gauze. Following removal of stitches, the tubes are prevented from falling out by transfixing the exposed ends with safety pins and fastening these to the skin with strips of sterilized adhesive tape (Fig. 6). Strips of vaseline gauze are placed over the margins of the wound to prevent gauze dressings from adhering to the wound. The tube drain is occasionally exchanged for a smaller one after the third week, but at no time is the cavity irrigated for fear of disseminating the infection. The patient is allowed to get out of bed after the second day and to leave the hospital after two weeks.

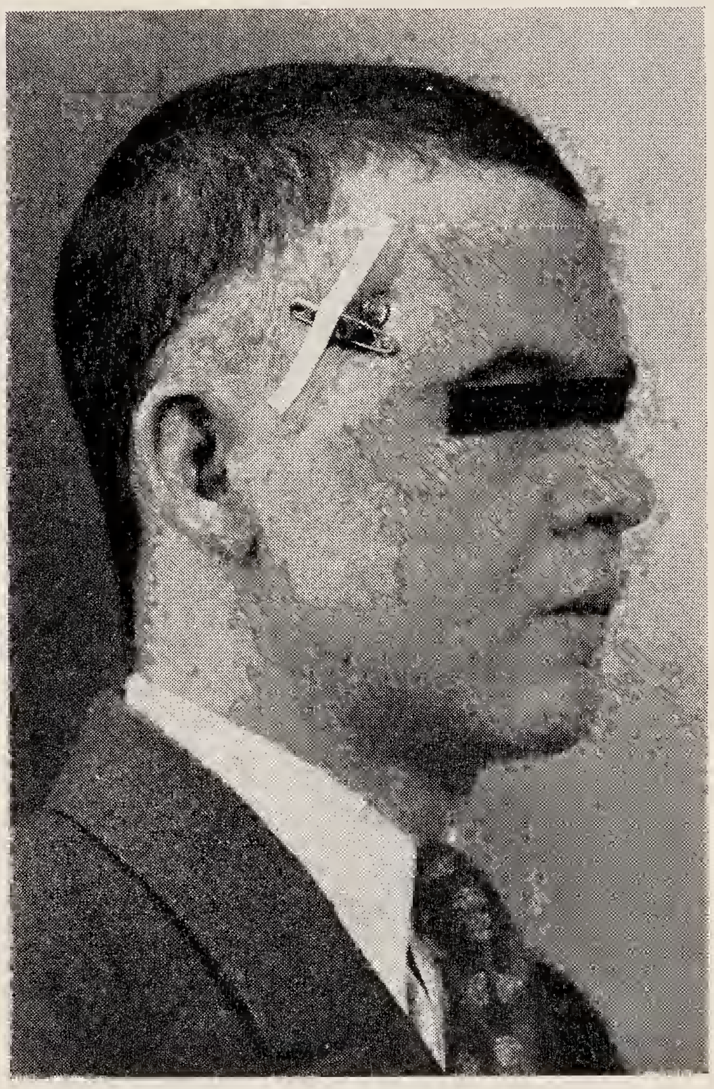

Fig. 6.—Appearance of wound, with one drainage tube in place, four weeks following drainage of an abscess of the right frontal lobe.

An abscess of the frontal lobe that is not associated with osteomyelitis of the frontal bone can be drained through a small opening placed back of the hair line through the frontal bone just above the zygoma. If osteomyelitis is present, one need not worry about additional scars and can then drain the abscess through the region of infection. We have found it safer to remove all suppurating and sequestrated bone before exploring for an abscess, unless there is evidence of an inflammatory zone extending from the dura into the brain. We prefer to cleanse the wound with tincture of iodine or merthiolate and to close it loosely and wait for the superficial infection to subside before proceeding with drainage of the abscess. If the inflammatory zone of the dura extends through into the brain, one might proceed with drainage at the initial operation, first by suturing and coagulating the meninges and cortex about the region to be explored. Instead of inserting the brain cannula blindly into the brain through the infected region, we prefer to incise the meninges and cortex with the electrosurgical needle to avoid contaminating normal brain tissue with the cannula. If the capsule is encountered, the usual procedure is employed; otherwise a gauze pack is placed into the cerebral wound to afford drainage while such further diagnostic measures as ventriculography are employed. The introduction of a cannula, knife, or needle into normal brain tissue during operation on the mastoid or frontal sinus or for osteomyelitis, is a dangerous procedure, since it will give rise to encephalitis and will cause more abscesses to be formed than would be accidentally found by such procedure.

Drainage by Aspiration.—Aspiration by needling has a limited field of usefulness. It is most useful in draining small, deep-seated abscesses. An aspiration or two may be sufficient to drain a sterile abscess, but when the organism still remains active, refilling continues until adequate, continuous drainage has been instituted. There is also danger of spreading the infection by repeated aspiration, as it is impossible to insert and withdraw the needle through the same tract.[13]

Drainage in Two Stages.—Dowman[14] and others have advocated operation in two stages for the purpose of allowing the brain to herniate through the opening in the skull before opening the abscess. With herniation, adhesions are supposed to be formed, preventing the cortex from separating from the wound margins and meninges when the abscess is evacuated at the second stage. Unfortunately this does not always occur, and contamination does take place during drainage. There is danger too that the brain may prolapse through the craniotomy opening, during the interval between the two stages, and give rise to an extensive cerebral fungus. An operation in two stages is most useful when it is necessary to treat active osteomyelitis before exploring for a cerebral abscess. In such circumstances the dura should not be opened until the second stage, and it should then be sealed to the cortex about the field to be explored with interrupted stitches of catgut and the electrocoagulation current in order to prevent any spread of the infection.

Treatment of the Capsule.—Again we find surgeons divided in their opinions as to the best treatment of the capsule. It must be accepted that if the capsule is left in place, it will result in a fibrous scar. Although this does occur, can it be avoided, and should the capsule be removed at the initial operation or allowed to remain unless symptoms of epilepsy appear? Macewen, Bagley, Hassin, Cone, and many others, have demonstrated the pathological changes that develop to form a capsule about the abscess. From our clinical experience it is apparent that the capsule continues to thicken for several months if the abscess goes unrecognized. Therefore, it is fair to assume that the walls will more readily collapse and be less thick if the abscess is properly drained as soon as immunity and encapsulation have taken place. To assure collapse of the capsule, King[25, 26] and Cahill[9] have suggested the removal of the overlying cortex and the peripheral dome of the capsule. King and others have suggested removal of the capsule at the initial operation. This procedure hastens recovery when it is possible to remove the abscess and capsule without opening it. Bagley reported such a case, and one of us (Adson) removed such an abscess but was unaware that he had done so until the mass was opened after removal. The wound in this case healed by primary intention, without any drainage. Our experience with removal of capsules following evacuation of the pus, however, has not been satisfactory, since a secondary suppurative encephalitis develops that is more troublesome to treat than the original infection. If drainage is not instituted, pus becomes inspissated and forms the caseous centre of a fibrous mass. The mass will gradually contract until there remains but a small nodule of what once was an abscess of 3 to 5 centimetres in diameter. The larger abscesses are more apt to rupture into the subarachnoid spaces and ventricular system than the smaller ones; consequently, it is unsafe to wait for these to disappear spontaneously. We cannot concur with those who believe that the rigid capsules will not collapse, for our experience has demonstrated that all have collapsed if properly drained. The difficulty encountered with recurrent abscesses, attributed to failure of the capsule to collapse, we believe is due to failure to secure adequate and continuous drainage, which has previously been referred to. Epilepsy develops too often following abscess, but a third of our patients complained of epileptiform seizures before operation. Hence, epilepsy is a symptom and a sequel that will be encountered in the treatment of suppurative diseases of the brain. Medication and diets offer some assistance in the management of epilepsy, but we are gradually becoming convinced that secondary operations, scar resections, as practiced by Penfield, are indicated in selected cases a year or later following the initial operation.

Temporal (via mastoid) Drainage of Abscess of the Temporal Lobe.—Abscesses that develop in the temporosphenoidal lobe are the result of extension of an infection from otitis media and mastoiditis with involvement of the antrum and tympanic wall. The infection may travel by contiguity, involving the meninges and brain over the inflamed bone, along bloodvessels, or through thrombosed infected veins. The latter is especially likely in the presence of a purulent thrombosis of the lateral sinus. Since the roof of the tympanic cavity is the thinnest of the communicating aural cavities, it represents the vulnerable spot where pus may first reach the dura. Extradural abscesses may appear without the development of a cerebral abscess. A serous meningitis often accompanies these phenomena, since the dura serves as an excellent screen to prevent sudden inward invasion of the organisms. However, if the virulence of the organism is too great, a septic meningitis with or without an accompanying encephalitis, develops. Frequently the meninges and cortex become so densely adherent to the diseased bone that only a localized meningitis with encephalitis develops, which gives rise to very mild meningitic symptoms, with low cell counts and no organisms in the cerebrospinal fluid. The particular situation of the abscess depends chiefly on the vein thrombosed and the depth to which the infection may travel. This undoubtedly explains why abscesses are found separated from their source of infection and vary in size and situation. Though the source of the infection may be the same, abscesses occur in the temporosphenoidal lobe more often than in the cerebellar lobes. Evans said that the ratio was

3:2, but our experience has indicated that the ratio has been 5:1, which is so high that when we are in doubt as to the situation of the abscess, we always explore the temporal lobe before exploring the cerebellum.

Since these abscesses arise from infections in or about the ear, the question is frequently raised as to whose province they belong, the otitic surgeon's or the nerosurgeon's. Fortunately this is not a serious question when coöperation between the two is maintained at all times. At the clinic, decision is based more on the type of abscess found. Following a radical mastoid operation the dura is carefully examined. If symptoms suggest cerebral involvement, it is determined whether or not the meninges and brain are adherent; if they are, the otitic surgeon incises the mass, extending the incision into the cortex in the hope of encountering an abscess which he then drains and treats. On the other hand if he exposes a clear arachnoid, he stops intracranial exploration and does not contaminate the brain by introducing an instrument from an infected mastoid wound into clean, normal, brain tissue. If symptoms continue, a separate craniotomy through a clean field is performed at a later date by one of the neurosurgeons. Whenever there is doubt as to the necessity for a mastoid operation as against craniotomy for cerebral abscess, the mastoid operation is performed first.

Relation of Frontal Sinusitis to Abscess of the Frontal Lobe.— Frontal sinusitis rarely gives rise to a cerebral abscess without being responsible for an accompanying osteomyelitis of the frontal bone. Woodward,[36] in considering the etiology and pathology of osteomyelitis of the frontal bone, has agreed with others that Staphylococcus pyogenes aureus is the organism most constantly responsible for the lesion, and he has stated that infection of the frontal sinus occludes the ostium and places the pus under pressure. This diminishes the blood supply, which in turn gives rise to necrosis of the mucous membrane and to thrombosis of the perforating veins. From this point the infection spreads through the diploë, preceded by thrombosis of diploic veins wherever death of bone has occurred. Pus and granulation tissue fill the diploë, which results in rapid destruction of the osseous elements. Furstenberg[18] has regarded the frontal sinus as eroded diploë, the outer wall corresponding to the external plates and the internal wall to the internal plates of the frontal sinus, which he believes is the chief reason for extension of the infection of the frontal sinus into the diploic spaces. Again we find the development of a cerebral abscess dependent on the circulation of the brain, for in ten of nineteen cases of frontal osteomyelitis observed in the Clinic abscesses were found in the frontal lobe. In the remainder the dura served as a barrier and prevented invasion of organisms, or the communicating veins had not thrombosed. The sinus infection is considered the emergency disease unless osteomyelitis of the frontal bone is fulminating in character, in which event both need immediate attention. Extradural abscesses over the frontal lobes are uncommon but they occur frequently enough to be watched for. Abscesses of the frontal lobe do develop from infections of the frontal sinus without an accompanying osteomyelitis, just as they develop from infection of the antrum or ethmoid and from infection of the maxilla following extraction of teeth.[27] These infections undoubtedly follow perivascular spaces and thrombosed veins which formerly drained, and extend into the white substance of the brain. Since these abscesses are usually single, it is not reasonable to expect the infection to be carried by the arterial circulation, for when it is, the embolic implants result in multiple abscesses. The treatment of this group of abscesses devolves on the neurosurgeon, since osteomyelitis may require an extensive cranial operation.

RELATION OF EXTENSIVE OSTEOMYELITIS TO CEREBRAL ABSCESS.—Osteomyelitis is considered the emergency disease since the infection will spread through diploë and emissary veins unless pressure is relieved. Small stab wounds are better than none, but if a cure is to be obtained, large flaps of scalp need to be reflected to remove thoroughly all suppurating and dead bone. In some instances only the outer table will be diseased, whereas in others the full thickness of the skull is involved. When this is true one should not hesitate to remove all the diseased bone, even though the dura is exposed; the wound should be cleansed with tincture of iodine and the scalp closed loosely, continuing with a similar area in three or four days. The procedure should be repeated until all the diseased bone has been removed. One can clean up a fourth of the skull area at one operation without undue surgical risk. After osteomyelitis has been brought under control, a separate incision may be made over the cerebral abscess and the abscess treated according to the technic described.

CEREBRAL ABSCESS OF TRAUMATIC ORIGIN.—*Preventive Treatment.*—The débridement procedure in compound comminuted fracture of the skull may readily forestall development of a cerebral abscess. It should be conducted with gentleness to avoid unnecessary injury to the brain and blood-vessels. Foreign particles, spicules of bone, blood-clots, and necrotic brain tissue should be removed by an aspirator. Prolapsed portions of the brain should be resected, and contaminated spicules of skull should be rongeured away. Necrotic margins of skin are removed before the edges are closed loosely with interrupted, non-absorbable sutures. Antiseptic solutions of iodine or merthiolate offer additional protection. Rubber-tissue drains can be used when indicated.

Puncture wounds lend themselves less to emergency treatment as they usually result from penetration of a sharp article through the orbit or nasal sinuses.

Débridement can be used for some gunshot wounds to advantage, but the ordinary bullet wound had better be left alone unless there is evidence of progressive bleeding, in which case emergency exploration is indicated.

Treatment by Drainage.—Such procedures are carried out very much as are those outlined under "surgical technic" except that exploration is usually made through the field of contamination whenever possible. The same principles of supportive treatment are employed as are used in the management of abscesses of the temporal and frontal lobes resulting from disease of the middle ear or from infections of the frontal sinus.

Cerebellar Abscess.—Cerebellar abscesses have been the most difficult to treat of all cerebral abscesses since collapse of the cerebellum following drainage usually results in contamination of the subarachnoid spaces and gives rise to a fatal meningitis.[10, 14, 15, 16] It is our treatment of this group of abscesses that has convinced us that the surgically sealed cerebral wound has a useful place in the treatment of brain abscesses. The usual preparation and delay is employed to make sure that encapsulation has taken place. It may be true that the zone of inflammation and adhesion is situated along the sigmoid sinus or petrous bone, but we have found it most advantageous to explore the cerebellum at the most accessible place, which is over the dorsum of each cerebellar lobe. A straight incision in the skin 3 centimetres to either side of the median line, will expose the occipital prominence. The technic from this point on is similar to that previously described. One cannot rely on electrocoagulation alone to seal the meninges and the cortex but must include numerous interrupted stitches of fine catgut to transfix the meninges and cortex around the margins of the proposed cerebellar exploration. There must be no leakage of cerebrospinal fluid. The cannula should be directed in an outward direction to avoid entering the fourth ventricle. Otherwise, the standard technic is used.

Pseudo-Abscess.—The term "pseudo" is used to designate a process that has progressed in a manner similar to the average cerebral abscess except that spontaneous recovery takes place instead of necrosis, liquefaction, and pus formation.[2] The avenues of infection are the same. The systemic reactions and the development of ophthalmological and neurological symptoms are not unlike those of abscess. Clinically the syndrome is similar to that of abscess.

Treatment should continue along supportive lines, but as long as a differential diagnosis cannot be made definitely without exploration, we have employed the routine surgical technic. On insertion of the cannula, resistance has been less marked than that of normal brain tissue, although at no time was pus obtained or a capsule encountered. Microscopical examination of aspirated material reveals some evidence of degeneration and infiltration of small, round cells and leucocytes. When the brain presented evidence of increased intracranial pressure, the small craniotomy has been enlarged, as for cerebral decompression, which has been most effective in decreasing choked disks. The failure of the localized encephalitis to become suppurative is due to a low grade of infection, to high immunity, or to a highly vascularized brain. We have encountered seven such cases and have performed exploratory operation in all of them without a fatality. All patients recovered except one who is occasionally troubled with an epileptiform seizure.

One additional case can be added in which there was circumscribed leptomeningitis with accumulation of purulent fluid in the sylvian fissure that produced all the signs and symptoms of abscess in the temporal lobe. Recovery following drainage in this case was prompt and permanent.

Doubtful Diagnosis.—All patients with symptoms suggesting brain abscess should be suspected of having a brain abscess and should be treated accordingly. Many of these patients recover without surgical intervention. Localized encephalitis clears up in some cases and small abscesses become fibrosed in others. Those patients who improve but fail to recover completely should be subjected to exploratory operation, for it is much safer to perform a small exploratory craniotomy than to let the patient continue with his symptoms, since large abscesses may rupture into the ventricular system or give rise to new extensions. In five instances we encountered a cerebral abscess when the history of infection was lacking and the patient was sent to operation with a diagnosis of cerebral neoplasm.

Review of Cases.—We have just completed a clinical and post-mortem review of cases in which patients were treated at The Mayo Clinic for cerebral abscess. Altogether ninety-seven patients were treated in the neurosurgical department by five different surgeons. Epidural abscesses about the petrous bone and those cerebral abscesses treated by the otitic surgeons are not included. Sixty patients were males, thirty-seven females. The average age was twenty-four years. Three patients were less than three months of age. Forty abscesses were situated in the frontal lobe, thirty-six in the temporosphenoidal, two in the occipital, and thirteen in the cerebellar. In the remaining six cases the abscesses were distributed miscellaneously.

The invading organisms were identified as Staphylococcus albus or aureus in nineteen cases, Streptococcus hæmolyticus in eleven, Streptococcus viridans in two, a combination of Streptococcus hæmolyticus and Streptococcus viridans in one, and as an unclassified form of streptococcus in five. There was a pneumococcal infection in one, pneumococci with diphtheroid bacilli in one, colon bacilli in one, streptococci with staphylococci in two, and bacilli of tuberculosis in one. In fifty-four cases laboratory reports were lacking or the culture was reported negative.

The review of chief complaints and past and present illnesses coincides with other similar reports. Headache was present in seventy-nine cases, nausea and vomiting in sixty-one, choked disks in forty-nine, and blindness in ten. Fifteen patients had pre-operative jacksonian convulsions and twenty had grand mal seizures. The neurological symptoms varied according to the size and position of the abscess as expected. The degree of recovery was in relation to the injury done by inflammation and surgical manipulation.

The results and mortality afforded an interesting study, as we could compare the handicraft of the various surgeons: 1. The results improved with the experience of the surgeon. 2. The mortality dropped as soon as the surgeon delayed drainage until encapsulation took place. 3. In abscesses of the temporosphenoidal lobes, there was a lower mortality when they were drained through a clean field in the temporal area than when they were drained through a suppurating mastoid wound. There is an occasional exception, but we believe that all would do better if drained independently of the mastoid wound. The same is true of cerebellar abscesses. 4. The sealing of the meninges and cortex about the drainage tract to prevent contamination has naturally improved convalescence and lowered the mortality. 5. Adequate continuous drainage with gauze packs and rubber-tube drains has prevented recurrent abscesses and cerebral herniation. These statements are substantiated by the recent experience of one of us who had but one death in fifteen consecutive cases of abscess situated above the tentorium, and but one death in three consecutive cases of abscess situated below the tentorium. The two patients who recovered had typical cerebellar abscesses and the one who died had an abscess that extended into the pons from the left cerebellar lobe.

SUMMARY

When cerebral abscesses follow infections about the ear, with indefinite localizing symptoms or with symptoms of conflicting signs of localization, we have observed the rule of exploring the temporosphenoidal lobe before exploring the cerebellum on the side of the infected ear because of the higher ratio of abscesses in the temporosphenoidal lobes than in the cerebellum.

In re-reading the protocols of Macewen's cases of cerebral abscess, we were impressed by the fact that in most of his cases there was a history of cerebral infection of a fortnight or more, which we believe is chiefly responsible for his excellent results, since in his cases sufficient time had elapsed for immunity and encapsulation to have been established before operation was undertaken.

Surgical drainage of an abscess does remove active organisms, but is probably more beneficial in disposing of the intracerebral mass. The arguments for delaying drainage of a cerebral abscess until it is mature, that is, encapsulated, which suggests that immunity has been established, are comparable to those for other inflammatory lesions of the body. One rarely advises drainage of an abdomen for acute peritonitis, although one urges adequate drainage when an abscess has been formed.

We believe, therefore, in administering supportive treatment during the initial stage of infection and in delaying drainage until clinical signs indicate that immunity has been established. We also prefer instituting adequate continuous drainage through a surgically-sealed cerebral wound to the various conservative or ultraradical measures employed.

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